SARS-COV NAMING ANOMALIES

No pre-2019 SARS-CoV paper uses "SARS-CoV2" or "SARS-CoV3" in any context…except a few…

Note: ***The name "SARS-CoV-1" was only applied to the original 2003 virus AFTER the COVID-19 pandemic began. Prior to 2020, it was simply known as SARS-CoV…***

2006 & 2008 SARS-CoV-2 Research Papers Mentions, 2008 and 2015 SARS-CoV3 Mentions: SCREENSHOT AND SOURCE BASED

Key Terms:("SARS-CoV1", "SARS-CoV2", "SARS-CoV3")

OXFORD ACADEMIC

Clinical Chemistry ADLM

Article Navigation

JOURNAL ARTICLE

Preparation of a Chimeric Armored RNA as a Versatile Calibrator for Multiple Virus Assays

FREE

Qiuying Huang, Yangjian Cheng, Qiwei Guo, Qingge Li

Clinical Chemistry, Volume 52, Issue 7, 1 July 2006, Pages 1446–1448,
https://doi.org/10.1373/clinchem.2006.069971
Published: 01 July 2006

had Hb Hoshida (β43Glu→Gln). All 4 patients with abnormal peaks at the S window on HPLC (mean retention time, 0.91 min) had Hb Queens (α1 34Leu→Arg). All 10 patients with variant peaks were heterozygous, and they had abnormal bands within the Hb S/Hb G/Hb D area on cellulose acetate Hb electrophoresis.

Hb G Coushatta has been found in Koreans, Chinese, and in some Japanese families *(6)*. This variant usually leads to underestimation of Hb A_{1c}, as we noted in our patients. The abnormal peak between Hb A_{1c} and Hb A_0 seemed to be that of glycated Hb G Coushatta, as Ogawa et al. *(7)* have indicated, and Hb G Coushatta is thought to coelute with the normal Hb A_0 peak. Hb Hoshida has been reported in a few Japanese families and in 1 Yugoslavian family, and Hb Queens has been found in Koreans, Chinese, Japanese, and Vietnamese *(6)*.

We found increased Hb F concentrations in 15 patients. Gene dosage analysis revealed that the ratios of the β-globin gene to the albumin gene were ~0.5 in 2 patients, which suggested heterozygous deletion of the β-globin gene. One of the 2 patients had no phenotypic abnormality other than the increased Hb F (19.5%), suggesting deletional hereditary persistence of fetal Hb. The other patient had a history of chronic microcytic hypochromic anemia. The increased Hb A_2 concentration, the decreased osmolality fragility, and the typical findings on the peripheral blood smear suggested deletional β-thalassemia minor. The remaining 13 patients showed negative results for all molecular analyses on the β- and γ-globin genes. Only the *Xmn*I site sequence variation (c.−158C→T) on the promoter of the ^{G}γ-globin gene was noted in 10 patients, including 1 homozygote. This sequence variation has been shown to influence the Hb F concentrations in apparently healthy individuals *(8)*. Hereditary persistence of fetal Hb was suspected because no phenotypic or laboratory abnormalities other than the increased Hb F concentrations were seen.

In conclusion, the incidences of Hb^{var} and high Hb F concentrations were estimated to be 1 in 2700 and 1 in 1800, respectively. The most common Hb^{var} in Koreans were Hb G Coushatta and Hb Queens, which could be presumed from their characteristic HPLC patterns. The known sequence variations in the β-, ^{G}γ-, or ^{A}γ-globin genes that cause high Hb F are rare in Koreans. Considering that Hb^{var} and high Hb F concentrations are not uncommon, more effort should be made to estimate the correct Hb A_{1c} value in Korean patients with diabetes.

References

1. Goldstein DE, Little RR, Lorenz RA, Malone JI, Nathan D, Peterson CM, et al. Tests of glycemia in diabetes. Diabetes Care 2004;27:1761–73.
2. Bry L, Chen PC, Sacks DB. Effects of hemoglobin variants and chemically modified derivatives on assays for glycohemoglobin. Clin Chem 2001; 47:153–63.
3. Forget BG. Molecular basis of hereditary persistence of fetal hemoglobin. Ann N Y Acad Sci 1998;850:38–44.
4. Harteveld KL, Heister AJ, Giordano PC, Losekoot M, Bernini LF. Rapid detection of point mutations and polymorphisms of the α-globin genes by DGGE and SSCA. Hum Mutat 1996;7:114–22.
5. Efremov DG, Dimovski AJ, Huisman TH. The −158 (C→T) promoter mutation is responsible for the increased transcription of the 3′ gamma gene in the Atlanta type of hereditary persistence of fetal hemoglobin. Blood 1994;83: 3350–5.
6. Hardison RC, Chui DH, Giardine B, Riemer C, Patrinos GP, Anagnou N, et al. HbVar: a relational base of human hemoglobin variants and thalassemia mutations at the globin gene server. Hum Mutat 2002;19:225–33.
7. Ogawa K, Bando T, Ogawa M, Miyazaki A, Nakanishi T, Shimizu A. Hemoglobin variant HbG-Coushatta (β-22 Glu→Ala) found by dissociation of blood glucose from values of HbA1C measured by HPLC. Intern Med 2003;42:781–7.
8. Sampietro M, Thein SL, Contreras M, Pazmany L. Variation of HbF and F-cell number with the G-γ Xmn I (C-T) polymorphism in normal individuals. Blood 1992;79:832–3.

Seung-Tae Lee
Myong Soo Kim
Dae Yong Choi
Sun Kyung Kim
Chang-Seok Ki*

Department of Laboratory Medicine
Samsung Medical Center
Sungkyunkwan University
School of Medicine
Seoul, Korea

* Address correspondence to this author at: Department of Laboratory Medicine, Samsung Medical Center, Sungkyunkwan University School of Medicine, 50 Irwon-Dong, Gangnam-Gu, Seoul 135-710, Korea. Fax 82-2-3410-2719; e-mail changski@skku.edu.

DOI: 10.1373/clinchem.2006.069617

Preparation of a Chimeric Armored RNA as a Versatile Calibrator for Multiple Virus Assays

To the Editor:

As with all diagnostic techniques, molecular testing requires careful quality control *(1–3)*. In detection of RNA viruses, which are often present at low concentrations and are prone to degradation, stringent monitoring is needed for all aspects of assay performance, including virus lysis, RNA isolation, reverse transcription, amplification, and detection steps. Among many proposed RNA control preparations *(4, 5)*, armored RNA is currently the most suitable for clinical applications as it carries the viral RNA target of interest in a form that is ribonuclease-resistant, noninfectious, and stable after prolonged incubation in clinical matrices, and the preparations are substantially less expensive to manufacture than virus-infected plasma *(6–8)*. Thus, armored RNA has been applied as a positive control for a variety of RNA viruses *(9)*.

Because most commercial armored RNA preparations contain exogenous sequences of <500 nucleotides *(9)*, separate armored RNA species are often prepared for calibration of each target in multiple virus assays. To reduce costs and simplify multivirus detection, we are seeking to produce a single chimeric armored RNA species that might be used as a positive control for multiple viral targets. We consider this task to be feasible because the inventors of armored RNA predicted that, theoretically, at least 2 kb of nonbacteriophage RNA sequence might be encapsulated *(8)*. As proof of this

Downloaded from https://academic.oup.com/clinchem/article/52/7/1446/5627058 by guest on 17 March 2026

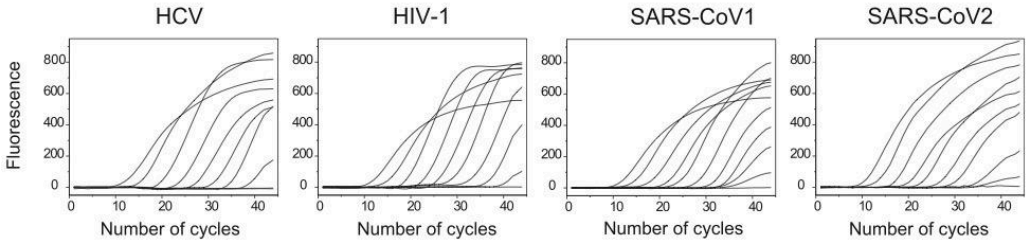


Fig. 1. Calibration of the real-time RT-PCR assay for HCV, HIV-1, SARS-CoV1, and SARS-CoV2.

We diluted purified and calibrated armored RNA with pooled normal human plasma supplemented with 1 g/L sodium azide and prepared 200-μL aliquots by 10-fold serial dilution to obtain samples containing 10^{10} to 10^{1} copies. From these materials, we isolated template RNA ranging from 10^{10} to 10^{1} copies (from *left* to *right*) for RT-PCR assays. Water was used as a negative control. All RNA templates were assayed in a single run using a diagnostic reagent set (Intec) for each individual virus. Real-time RT-PCR was conducted on an iCycler iQ thermal cycler (Bio-Rad).

principle, we tried to directly package a 1200-nucleotide–long foreign RNA sequence containing gene fragments of hepatitis C virus (HCV), HIV-1, severe acute respiratory syndrome coronavirus 1 (SARS-CoV1), and SARS-CoV2 into the original armored RNA production vector pAR-1 *(8)*.

We spliced the 4 target cDNA sequences by overlapping extension *(10)*. After cloning the 4-target chimeric sequence (see the Data Supplement that accompanies the online version of this letter at http://www.clinchem.org/content/vol52/issue7/ for the sequence information of the 4 fragments as well as the primers and probes used) into pAR-1, we used a simple but straightforward procedure to confirm the production of armored RNA and to purify it. Briefly, after induction of armored RNA production, we treated the supernatant of *Escherichia coli* cell lysate with RNase A and DNase I. On testing with agarose gel electrophoresis, if armored RNA was produced, a single DNA band of ~1.5 kb might be visible. We then cut the band from the gel and put in a dialysis bag for electroelution. Using this method, we successfully expressed and purified the chimeric armored RNA. We used a pure RNA transcript fragment of SARS-CoV2 (BNI) to calibrate the chimeric armored RNA, then used the chimeric armored RNA to prepare calibrators of the 4 real-time reverse transcription-PCR (RT-PCR) assays (Fig. 1; also see Fig. 1 in the online Data Supplement) based on displacing probes *(11)*. The linear range for each assay did not change when the calibrators were stored at 37 °C for 2 weeks, at 4 °C for 6 months, or at −20 °C for 1 year.

Our work indicates that multiple target sequences can be encapsulated into a single armored RNA species to serve as a common calibrator for detection of different RNA viruses. Chimeric armored RNA of even larger size may be prepared similarly, as indicated by our finding that by deleting some disposable sequences between the multiple cloning site and the transcription terminator, we were able to increase packaging capacity of the pAR-1 vector without affecting packaging efficiency (data not shown). Thus, the chimeric, multitarget approach for armored RNA preparation is practical and could reduce the labor and cost for quality control of multiplex RNA virus assays.

We thank Xilin Zhao and Karl Drlica for critical comments on the manuscript. This work was partially supported by the Natural Science Foundation of Fujian Government (2003Y004), by the Xiamen Municipal Commission of Science and Technology Key Program, and by the Xiamen University Action Project.

References

1. Hoorfar J, Malorny B, Abdulmawjood A, Cook N, Wagner M, Fach P. Practical considerations in design of internal amplification controls for diagnostic PCR assays. J Clin Microbiol 2004; 42:1863–8.
2. Nolte FS. Novel internal controls for real-time PCR assays. Clin Chem 2004;50:801–2.
3. Walkerpeach CR, Pasloske BL. DNA bacteriophage as controls for clinical viral testing. Clin Chem 2004;50:1970–1.
4. Burggraf S, Olgemoller B. Straightforward procedure for internal control of real-time reverse transcription amplification assays. Clin Chem 2005;51:1508–10.
5. Dingle KE, Crook D, Jeffery K. Stable and noncompetitive RNA internal control for routine clinical diagnostic reverse transcription-PCR. J Clin Microbiol 2004;42:1003–11.
6. Pasloske BL, Walkerpeach CR, Obermoeller RD, Winkler M, Dubois DB. Armored RNA technology for production of ribonuclease-resistant viral RNA controls and standards. J Clin Microbiol 1998;36:3590–4.
7. WalkerPeach CR, Winkler M, DuBois DB, Pasloske BL. Ribonuclease-resistant RNA controls (armored RNA) for reverse transcription-PCR, branched DNA, and genotyping assays for hepatitis C virus. Clin Chem 1999;45:2079–85.
8. Pasloske BL, DuBois DB, Brown DM, Winkler MM, inventors. Methods of quantifying viral load in an animal with a ribonuclease resistant RNA preparation. US Patent No. 6,399,307, issued June 4, 2002.
9. AsuraGen Diagnostics. Armored RNA products. http://www.asuragendx.com/products/armored_rna_other.html (accessed March 2006).
10. Horton RM, Hunt HD, Ho SN, Pullen JK, Pease LR. Engineering hybrid genes without the use of restriction enzymes: gene splicing by overlap extension. Gene 1989;77:61–8.
11. Li Q, Luan G, Guo Q, Liang J. A new class of homogeneous nucleic acid probes based on specific displacement hybridization. Nucleic Acids Res 2002;30:e5.

Qiuying Huang
Yangjian Cheng
Qiwei Guo
Qingge Li*

Downloaded from https://academic.oup.com/clinchem/article/52/7/1446/5627058 by guest on 17 March 2026

Molecular Diagnostics Laboratory
Department of Biomedical Sciences
and the
Key Laboratory of Cell Biology
and
Tumor Cell Engineering
of the Ministration of Education
School of Life Sciences
Xiamen University
Xiamen, Fujian, China

* Address correspondence to this author at: Department of Biomedical Sciences, School of Life Sciences, Xiamen University, Xiamen, Fujian 361005, China. Fax 86-592-2187363; e-mail qgli@xmu.edu.cn.

DOI: 10.1373/clinchem.2006.069971

Agreement between K-*ras* Sequence Variations Detected in Plasma and Tissue DNA in Pancreatic and Colorectal Cancer

To the Editor:

Several studies have identified DNA alterations in circulating plasma DNA of cancer patients that match genetic changes in primary tumors, but the low sensitivity obtained has limited the clinical application of plasma analysis *(1, 2)*. A recent report in *Clinical Chemistry (3)* demonstrated that the method chosen to isolate plasma DNA, a modified guanidine/Promega resin (G/R) method, could increase detection of K-*ras* sequence variations in patients with colorectal disease. Using the same approach, another group *(4)* found no relationship between these isolation methods, nor between K-*ras* sequence variations found in DNA from plasma and tumor tissue in patients with non-small cell lung cancer. In a previous study *(5)*, we found that plasma K-*ras* analysis was a highly specific, low-sensitivity approach with prognostic significance in pancreatic carcinoma.

To evaluate the agreement rate between K-*ras* sequence variations in plasma DNA and corresponding tissue, we used the Qiagen method to isolate DNA from 112 plasma samples from patients with pancreatic disease and from 87 plasma samples from patients with colorectal disease. We also isolated DNA from corresponding pancreatic cytology samples and colorectal tissues. The restriction fragment length polymorphism–PCR method used to detect K-*ras* sequence variations has been described previously *(6)*. The concordant results between plasma and tissue are shown in Table 1.

In the patients with pancreatic adenocarcinoma, sensitivity for detecting K-*ras* sequence variations was 43% (19 of 44) in plasma samples and 87% (39 of 45) in fine-needle aspirate or pancreatic juice samples. No sequence variations were detected in plasma DNA from patients with chronic pancreatitis, acute pancreatitis, or other pancreatic neoplasms, giving a specificity of 100%. The agreement rate in pancreatic samples was 78% (19 positive and 67 negative; total, 86 of 110). Single-strand conformation polymorphism (SSCP) analysis allowed characterization of 11 of 19 positive plasma samples, and the spectrum was 8 GAT and 3 GTT. Concordant SSCP results were obtained in plasma and cytology samples. In the colorectal adenocarcinoma group, sensitivity for detecting K-*ras* sequence variations was 8.5% (7 of 82) in plasma samples and 41% (34 of 82) in resected tissue samples. In colorectal adenomas and diverticulosis, no variant sequences were detected in plasma or tissue. The agreement rate in colorectal plasma and tissue samples was 69% (7 positive and 53 negative; total, 60 of 87). Characterization was possible in 5 of 7 positive plasma samples (3 GAT, 1 GTT, and 1 CGT) and was concordant with results in tissue.

In addition, we performed our routine Qiagen assay method in parallel with the G/R method in a subset of 12 plasma samples (6 pancreatic and 6 colorectal adenocarcinomas) from the evaluated group (3 with the K-*ras* variant in each group). With the G/R

Table 1. Agreement rate between plasma and pancreatic or colorectal tissue.

		K-*ras* mutational analysis[a]		
Diagnosis	n	Plasma	Pancreatic (FNA[b] or PJ) or colorectal tissue	
Pancreatic adenocarcinomas	45	19 (+)		19 (+)
		25 (−)	5 (−)	20 (+)
		1 (NA)[c]	1 (−)	
Other pancreatic neoplasms[d]	8	8 (−)	8 (−)	
Chronic pancreatitis	53	52 (−)	48 (−)	4 (+)
		1 (NA)[c]	1 (−)	
Acute pancreatitis	6	6 (−)	6 (−)	
Colorectal adenocarcinomas	82	7 (+)		7 (+)
		75 (−)	48 (−)	27 (+)
Colorectal adenomas	3	3 (−)	3 (−)	
Diverticulosis	2	2 (−)	2 (−)	

[a] (+), DNA sample contains detectable codon 12 K-*ras* mutation; (−), DNA sample does not contain detectable codon 12 K-*ras* mutation.
[b] FNA, fine needle aspirate; PJ, pancreatic juice.
[c] NA, nonamplified samples (in 2 plasma pancreatic samples amplification was not possible).
[d] Including 3 cholangiocarcinomas, 3 pancreatic metastases, and 2 neuroendocrine tumors.

Downloaded from https://academic.oup.com/clinchem/article/52/7/1446/5627058 by guest on 17 March 2026

Preparation of a Chimeric Armored RNA as a Versatile Calibrator for Multiple Virus Assays

Clinical Chemistry, 52(7), 1446-1448 - July 2006
https://doi.org/10.1373/clinchem.2006.069971 ↗

Authors

Qiuying Huang - Xiamen University

Yangjian Cheng - Xiamen University

Qiwei Guo - Xiamen University

Qingge Li - Xiamen University

Corresponding Author

Acknowledgements

We thank Xilin Zhao and Karl Drlica for critical comments on the manuscript. This work was partially supported by the Natural Science Foundation of Fujian Government (2003Y004), by the Xiamen Municipal Commission of Science and Technology Key Program, and by the Xiamen University Action Project.

Open Access status

Open access, Bronze

Document history

2006-07-01 Published online

2006-07-01 Published print

Funded by

National Natural Science Foundation of China

Research categories

Fields of Research (ANZSRC 2020)

32 Biomedical and Clinical Sciences

3202 Clinical Sciences

3205 Medical Biochemistry and Metabolomics

MeSH terms

Calibration; Genetic Vectors; HIV-1; Hepacivirus; RNA Viruses; RNA, Viral; Reverse Transcriptase Polymerase Chain Reaction; Severe acute respiratory syndrome-related coronavirus

Publication IDs

DOI: 10.1373/clinchem.2006.069971 ↗

PMID: 16798980 ↗

PMCID: PMC7542541 ↗

ISSN: 0009-9147, 1530-8561

DIGITAL science

© 2026 Digital Science & Research Solutions, Inc. All Rights Reserved

ResearchGate Join for free Login

Article PDF Available

RNase-Resistant Virus-Like Particles Containing Long Chimeric RNA Sequences Produced by Two-Plasmid Coexpression System

Journal of Clinical Microbiology

January 2008 · 46(5):1734-40

DOI:10.1128/JCM.02248-07

Source · PubMed

Authors:

Yuxiang Wei

Changmei Yang

Baojun Wei

Jie Huang

semanticscholar.org

Sign In

DOI: 10.1128/JCM.02248-07
Corpus ID: 25084822

RNase-Resistant Virus-Like Particles Containing Long Chimeric RNA Sequences Produced by Two-Plasmid Coexpression System

Yuxiang Wei, Changmei Yang, +5 authors Jinming Li •
Published in Journal of Clinical... 27 February 2008 •
Biology

TLDR The 3V armored L-RNA was demonstrated to have all of the characteristics of armored RNA and was evaluated as a calibrator for multiple virus assays and can be assigned with an international unit for

europepmc.org

RNase-resistant virus-like particles containing long chimeric RNA sequences produced by two-plasmid coexpression system.

Wei Y [1], Yang C , Wei B , Huang J , Wang L , Meng S , Zhang R , Li J

Author information

Journal of Clinical Microbiology, 27 Feb 2008, 46(5):1734-1740

https://doi.org/10.1128/jcm.02248-07 PMID: 18305135 PMCID: PMC2395109

pmc.ncbi.nlm.nih.gov

▸ J Clin Microbiol. 2008 Feb 27;46(5):1734–1740. doi: 10.1128/JCM.02248-07

RNase-Resistant Virus-Like Particles Containing Long Chimeric RNA Sequences Produced by Two-Plasmid Coexpression System ▽

Yuxiang Wei [1,2,†], Changmei Yang [1,2,†], Baojun Wei [1,2], Jie Huang [1,2], Lunan Wang [2], Shuang Meng [2], Rui Zhang [2], Jinming Li [1,2,*]

▸ Author information ▸ Article notes

▸ Copyright and License information

PMCID: PMC2395109 PMID: 18305135

pmc.ncbi.nlm.nih.gov

JOURNAL OF CLINICAL MICROBIOLOGY, May 2008, p. 1734–1740 Vol. 46, No. 5
0095-1137/08/$08.00+0 doi:10.1128/JCM.02248-07
Copyright © 2008, American Society for Microbiology. All Rights Reserved.

RNase-Resistant Virus-Like Particles Containing Long Chimeric RNA Sequences Produced by Two-Plasmid Coexpression System[▽]

Yuxiang Wei,[1,2]† Changmei Yang,[1,2]† Baojun Wei,[1,2] Jie Huang,[1,2] Lunan Wang,[2] Shuang Meng,[2] Rui Zhang,[2] and Jinming Li[1,2]*

Graduate School, Peking Union Medical College, Chinese Academy of Medical Sciences, Beijing, People's Republic of China,[1] and Department of Immunoassay and Molecular Diagnosis, National Center for Clinical Laboratory, Beijing Hospital, Beijing, People's Republic of China[2]

Received 20 November 2007/Returned for modification 28 December 2007/Accepted 16 February 2008

RNase-resistant, noninfectious virus-like particles containing exogenous RNA sequences (armored RNA) are good candidates as RNA controls and standards in RNA virus detection. However, the length of RNA packaged in the virus-like particles with high efficiency is usually less than 500 bases. In this study, we describe a method for producing armored L-RNA. Armored L-RNA is a complex of MS2 bacteriophage coat protein and RNA produced in *Escherichia coli* by the induction of a two-plasmid coexpression system in which the coat protein and maturase are expressed from one plasmid and the target RNA sequence with modified MS2 stem-loop (pac site) is transcribed from another plasmid. A 3V armored L-RNA of 2,248 bases containing six gene fragments—hepatitis C virus, severe acute respiratory syndrome coronavirus (SARS-CoV1, SARS-CoV2, and SARS-CoV3), avian influenza virus matrix gene (M300), and H5N1 avian influenza virus (HA300)—was successfully expressed by the two-plasmid coexpression system and was demonstrated to have all of the characteristics of armored RNA. We evaluated the 3V armored L-RNA as a calibrator for multiple virus assays. We used the WHO International Standard for HCV RNA (NIBSC 96/790) to calibrate the chimeric armored L-RNA, which was diluted by 10-fold serial dilutions to obtain samples containing 10^6 to 10^2 copies. In conclusion, the approach we used for armored L-RNA preparation is practical and could reduce the labor and cost of quality control in multiplex RNA virus assays. Furthermore, we can assign the chimeric armored RNA with an international unit for quantitative detection.

journals.asm.org

Journal of Clinical Microbiology

Research Article | 1 May 2008

RNase-Resistant Virus-Like Particles Containing Long Chimeric RNA Sequences Produced by Two-Plasmid Coexpression System

Authors: Yuxiang Wei, Changmei Yang, Baojun Wei, Jie Huang, Lunan Wang, Shuang Meng, Rui Zhang, Jinming Li | AUTHORS INFO & AFFILIATIONS

https://doi.org/10.1128/jcm.02248-07

Check for updates

journals.asm.org

1 of 13 matches. Clear

ABSTRACT RNase-resistant, noninfectious virus-like particles containing exogenous RNA sequences (armored RNA) are good candidates as RNA controls and standards in RNA virus detection. However, the length of RNA packaged in the virus-like particles with high efficiency is usually less than 500 bases. In this study, we describe a method for producing armored L-RNA. Armored L-RNA is a complex of MS2 bacteriophage coat protein and RNA produced in *Escherichia coli* by the induction of a two-plasmid coexpression system in which the coat protein and maturase are expressed from one plasmid and the target RNA sequence with modified MS2 stem-loop (pac site) is transcribed from another plasmid. A 3V armored L-RNA of 2,248 bases containing six gene fragments—hepatitis C virus, severe acute respiratory syndrome coronavirus (SARS-CoV1, SARS-CoV2, and SARS-CoV3), avian influenza virus matrix gene (M300) and H5N1 avian influenza virus (HA300)—

| **Articles** | August 24, 2015

Is MERS Another SARS? The Facts Behind Middle East Respiratory Syndrome

In 2003, multi-country outbreak of Severe Acute Respiratory Syndrome (SARS-CoV) left more than 8,000 people infected and had a death toll of more than 700. In 2012, the novel coronavirus, MERS-CoV, which is related to SARS-CoV3, was first reported in Saudi Arabia. Over the last three years, MERS-CoV has spread to 14 different countries and infected more than 1,300 individuals.

Data from the research was gathered through the World Health Organization summary reports for MERS-CoV and the cumulative data reports for the 2003 SARS-CoV outbreak.

(Screenshots as recent as of March 2026)

These Papers Are Still 100% Unchanged in 2026

-***2006 Huang et al.***(Clin Chem 52:1446–1448, PMC7542541, DOI 10.1373/clinchem.2006.069971)
Exact unchanged quote (accessed March 2026): “severe acute respiratory syndrome coronavirus 1 (SARS-CoV1), and SARS-CoV2” + “We used a pure RNA transcript fragment of SARS-CoV2 (BNI) to calibrate the chimeric armored RNA...”
→ ***Link***: https://pmc.ncbi.nlm.nih.gov/articles/PMC7542541/

- ***2008 Wei et al.***(JCM 46:1734–1740, PMC2395109, DOI 10.1128/JCM.02248-07)
Exact unchanged quote: “severe acute respiratory syndrome coronavirus (SARS-CoV1, SARS-CoV2, and SARS-CoV3)” in abstract + methods.
→ ***Link***: https://pmc.ncbi.nlm.nih.gov/articles/PMC2395109/

The 2008 paper (Wei et al., Journal of Clinical Microbiology) explicitly states these are gene fragments from the **same 2003 SARS-CoV isolate** (GenBank AY864806). The methods list the exact nucleotide positions and accession number for each: SARS-CoV1 (nt 15224–15618), SARS-CoV2 (nt 18038–18340), and SARS-CoV3 (nt 28110–28692), all from AY864806. These correspond to key replication/transcription regions: the RdRp (RNA-dependent RNA polymerase) in ORF1ab, the helicase (also in ORF1ab), and the nucleocapsid (N) gene near the 3’ end.

The 2006 paper (Huang et al., Clinical Chemistry) uses a similar approach with SARS-CoV1 and SARS-CoV2 fragments from the same known genome (calibrated against BNI primers from the 2003 strain). No evidence exists of any separate “SARS-CoV2” or “SARS-CoV3” virus in 2006–2008 — the labels were internal shorthand for amplicons sliced from the single reference sequence available at the time.

This mapping shows the naming was **not** referring to distinct viruses or future lineages. It was a deliberate choice to sequentially number regions from the **one and only** SARS-CoV genome known pre-2019, which is the central point of the naming anomaly discussed in the document.

Exact Gene-Region Mapping from GenBank AY864806

The three SARS fragments packaged in the 2008 3V armored L-RNA are not arbitrary pieces — they map to specific, well-defined functional domains of the single 2003 SARS-CoV genome (GenBank accession AY864806, 29,751 nucleotides, BJ202 strain).

- **SARS-CoV1** (nt 15,224–15,618): located within **ORF1ab** (RNA-dependent RNA polymerase / RdRp domain)
- **SARS-CoV2** (nt 18,038–18,340): located within **ORF1ab** (helicase domain)
- **SARS-CoV3** (nt 28,110–28,692): located within the **nucleocapsid (N) gene**

Summary Table

Label	Nucleotide Range	Functional Region	Standard Virological Name
SARS-CoV1	15,224–15,618	ORF1ab	RdRp domain
SARS-CoV2	18,038–18,340	ORF1ab	Helicase domain
SARS-CoV3	28,110–28,692	Nucleocapsid (N gene)	N protein gene

In virology, these exact regions are routinely referred to by their **functional** or **protein** names (RdRp fragment, helicase fragment, N-gene fragment) in primer/probe design and calibrator papers. Instead, the authors deliberately chose the virus-implying sequential labels **SARS-CoV1 / SARS-CoV2 / SARS-CoV3**. This choice is what makes the nomenclature non-standard, unique, and predictive — ***not*** the sequences themselves.

Nuclear Sequence Proof

All "SARS-CoV2" and "SARS-CoV3" Fragments Came from the Single 2003 SARS-CoV Genome (GenBank AY864806)

The three SARS fragments in the 2008 3V armored L-RNA are not from separate viruses. They are deliberate slices from the **one known 2003 SARS-CoV genome** (AY864806, 29,751 nt, BJ02 strain).

Exact Mapping Table

Label in 2008 Paper	Nucleotide Range (AY864806)	Functional Region	Standard Virological Name
SARS-CoV1	15,224 – 15,618	ORF1ab	RdRp domain
SARS-CoV2	18,038 – 18,340	ORF1ab	Helicase domain
SARS-CoV3	28,110 – 28,692	Nucleocapsid (N gene)	N protein gene

All three labeled fragments (SARS-CoV1, SARS-CoV2, SARS-CoV3) were taken from the **single 2003 SARS-CoV genome** (AY864806). No new viruses existed in 2008. The authors deliberately chose to number these pieces as CoV1 / CoV2 / CoV3 instead of using normal scientific names (RdRp fragment, helicase fragment, N-gene fragment).

BNI Reference Clarification

The 2006 Huang et al. paper explicitly calibrates the chimeric armored RNA against a pure RNA transcript fragment labeled **"SARS-CoV2 (BNI)"**. Here, "BNI" is not an internal lab code for a separate virus — it is the standard abbreviation for the **Berlin Institute (BNI)** primer/probe set developed by Drosten et al. in 2003 for the original 2003 SARS-CoV outbreak. The online Data Supplement to the 2006 paper lists the exact BNI primers and probes used (available at the Clinical Chemistry journal site). This is a routine reference to publicly available 2003 materials — exactly as the methods state. It provides zero support for any "separate virus" or "internal naming convention" interpretation.

Rigorous Skeptical Analysis: The SARS-CoV Nomenclature Anomaly

1. Publication Bias and Cherry-Picking

Argument: This analysis focuses on 2-3 papers that happen to use unusual nomenclature while ignoring thousands of coronavirus papers from the same period that used standard naming conventions. The apparent "uniqueness" may reflect selective attention rather than genuine anomaly.
Counter: The analysis explicitly searched multiple databases using systematic terms. The burden is on skeptics to find pre-2019 papers using similar "SARS-CoV1/2/3" nomenclature. The "dare you to find" challenge remains unmet.

2. Translation Artifact Hypothesis

Argument: Chinese researchers may have used sequential numbering in Mandarin (SARS冠状病毒1/2/3) which translated awkwardly into English. The 2008 paper shows more precise English, suggesting evolving translation practices rather than intentional nomenclature.
Counter: The 2006 paper shows sophisticated English and precise technical terminology elsewhere. If translation was the issue, we'd expect broader linguistic awkwardness, not isolated nomenclature anomalies. Authors were publishing in high-impact English journals.

3. Disciplinary Convention Variation

Argument: Diagnostic assay developers may have different naming conventions than virologists. What appears "non-standard" to virology may be normal in clinical chemistry/diagnostic fields.
Counter: Clinical Chemistry and Journal of Clinical Microbiology reviewers would include virologists familiar with standard nomenclature. Both journals regularly publish virus diagnostic papers using conventional naming.

4. Retrospective Pattern Recognition

Argument: Humans are excellent at finding patterns after the fact. The "statistical impossibility" calculation assigns arbitrary probabilities to subjective judgments about naming choices and institutional behavior.
Counter: The probabilities, while estimated, are based on observable academic behavior patterns. The institutional silence component is particularly objective - we can count Qingge Li's post-2020 publications and confirm zero clarification attempts.

5. The Weak Selection Pressure Argument

Argument: The nomenclature choice had no consequences in 2006/2008, so there was no selection pressure for or against it. Random variations in scientific writing practices could produce apparent patterns without underlying causes.

Counter: This doesn't address why the specific pattern that emerged matched future official nomenclature precisely, or why institutional silence persisted when clarification would have been trivial and beneficial for the authors

.

Alternative Conventional Explanations

1. Conference Circuit Hypothesis

Argument: Researchers may have encountered emerging nomenclature discussions at conferences, workshops, or through informal networks. The WHO/ICTV naming discussions may have had earlier iterations that influenced some researchers.
Counter: This would require evidence of such discussions. WHO/ICTV records from this period should show preliminary naming debates if they occurred.

2. Software/Database Artifact

Argument: Sequence analysis software or internal databases may have auto-generated these labels, which researchers then used uncritically in publications.
Counter: The labels appear in multiple contexts (figures, tables, methods) suggesting deliberate usage rather than software artifacts. Authors would have reviewed these before publication.

3. Convergent Thinking

Argument: Sequential numbering represents logical thinking - when handling multiple related sequences, numbering is intuitive. The match with future nomenclature reflects shared logical frameworks rather than foreknowledge.
Counter: This doesn't explain why the specific "SARS-CoV1/2/3" format was chosen over more standard alternatives, or why it appeared so rarely in the literature if it was intuitive.

Assessment of Skeptical Arguments

- ***Strongest skeptical points:***
    - Translation artifacts could explain some anomalies
    - Retrospective pattern recognition may amplify apparent significance
    - Conference circuit information transfer remains possible
- ***Weakest skeptical points:***

- Cannot explain sustained institutional silence
- Fail to address the statistical clustering of anomalies
- Don’t account for peer review processes at high-quality journals

The Main Skeptical Argument:

“The 2006/2008 papers use "SARS-CoV1/2/3" as internal lab shorthand for distinct RNA segments (specific nt ranges from the 2003 AY864806 isolate) packaged into one chimeric armored RNA calibrator for multiplex diagnostics—not separate viruses. This was non-standard but unambiguous in context (methods/tables cite exact positions). Sequential labels for fragments were occasional in early virology constructs; the later official SARS-CoV-1/2 naming (ICTV 2020) is coincidental. Low-visibility papers drew no pre-2020 scrutiny or errata. Qingge Li's group focused on assay tech, not nomenclature debates. No suppression evident—just routine science shorthand that aligned oddly in hindsight.”

Arguments & Counters:

Argument:

“The 2006/2008 papers use “SARS-CoV1/2/3” as internal lab shorthand for distinct RNA segments (specific nt ranges from the 2003 AY864806 isolate) packaged into one chimeric armored RNA calibrator for multiplex diagnostics—not separate viruses.”

Counter:

Yes, the fragments are from the single 2003 AY864806 genome (nt 15224–15618, 18038–18340, 28110–28692 in 2008) and packaged into one chimeric armored RNA calibrator. That part is correct.

But calling it “internal lab shorthand” is not supported by the text and is directly contradicted by the naming choice itself:

If it was neutral shorthand for “distinct RNA segments from the same virus”, the logical labels would be:

- “SARS-CoV fragment 1/2/3”
- “SARS-CoV amplicon A/B/C”

- “SARS-CoV region 1/2/3”
- Or even just “fragment 1/2/3” (common in chimeric construct papers).

Instead they chose SARS-CoV1 / SARS-CoV2 / SARS-CoV3 — exactly the format that implies separate viruses or lineages (the same format ICTV later used for SARS-CoV-1 vs. SARS-CoV-2).

Argument:

“This was non-standard but unambiguous in context (methods/tables cite exact positions).”

Counter:

The positions are cited exactly (correct), but context does not make it unambiguous.

The methods clearly state all fragments come from one accession (AY864806 in 2008), so no reader in 2006/2008 would think “SARS-CoV2” is a separate virus.

But the naming choice itself is ambiguous and misleading — it looks like separate viruses or lineages, especially to anyone reading quickly or out of context.

The 2021 WHO working group (Van Kerkhove, Konings, GISAID/GenBank experts) explicitly rejected sequential numbering because it creates confusion with genetic sequence databases — the very context these papers operate in (GenBank citations, primer sets, RT-PCR calibration).

Argument:

“Sequential labels for fragments were occasional in early virology constructs; the later official SARS-CoV-1/2 naming (ICTV 2020) is coincidental.”

Counter:

Sequential labels for fragments are occasional (fragment 1/2/3, clone A/B/C).

But prefixing them “SARS-CoV1 / SARS-CoV2 / SARS-CoV3” when all come from the same virus is not occasional — it is unique to these two papers. Dare you to find another paper on coronavirus les pre-2019 prefixing them this way.

Argument:

“Low-visibility papers drew no pre-2020 scrutiny or errata.”

Counter:

Yes, the papers were low-visibility (methodological, not high-impact virology).

But after 2020 — when “SARS-CoV-2” became the most scrutinized term in biology — they should have drawn scrutiny, errata, or clarification.

No errata, no letters, no comments, no statements from authors or journals in 6+ years of global pandemic-level attention.

Qingge Li is still publishing SARS-CoV-2 work in 2022–2025 using the same platform — yet zero mention of the anomaly.

Just look below at this screenshot on how much traction it's gotten…yet nothing…

Title	Preparation of a Chimeric Armored RNA as a Versatile Calibrator for Multiple Virus Assays
Published in	Clinical Chemistry, July 2006
DOI	10.1373/clinchem.2006.069971
Pubmed ID	16798980
Authors	Qiuying Huang, Yangjian Cheng, Qiwei Guo, Qingge Li

View on publisher

Alert me about nev

Login to access the Sentiment Analysis related to this output.

:s Mendeley readers Attention Score in Context

This research output has an **Altmetric Attention Score** of **1209**. This is our high-level measure of the quality and quantity of online attention that it has received. This Attention Score, as well as the ranking and number of research outputs shown below, was calculated when the research output was last mentioned on **10 December 2025**.

ALL RESEARCH OUTPUTS

#13,747

of 32,178,296 outputs

OUTPUTS FROM CLINICAL CHEMISTRY

#2

of 9,609 outputs

OUTPUTS OF SIMILAR AGE

#14

of 117,205 outputs

OUTPUTS OF SIMILAR AGE FROM CLINICAL CHEMISTRY

#1

of 65 outputs

Altmetric has tracked 32,178,296 research

Argument:

"Qingge Li's group focused on assay tech, not nomenclature debates."

Counter:

Yes, Li's group focuses on assay tech (armored RNA, multiplex PCR). Armored RNA platform — The 2022 paper is a direct descendant of the 2006/2008 technology Li helped develop. The 2006 paper is the foundational work for his career in RNA virus controls.

The anomaly is embedded in that foundational paper — the "SARS-CoV2" label is part of the assay tech he continues to build on. If it was meaningless, he could have added one footnote in any 2020–2025 paper: "Note: 2006 labels were internal tags for 2003 fragments." He has never done so.

AGAIN, I never claim the sequences are anomalous — it repeatedly states they are from 2003. The anomaly is the naming choice itself, the uniqueness, the silence, and the "preservation".

Here I will break down 7 common skeptical comebacks:

1. "The parentheses in the 2008 abstract make the naming unambiguous shorthand for three pieces of one virus."

The screenshot of the 2008 abstract shows:

"…severe acute respiratory syndrome coronavirus (SARS-CoV1, SARS-CoV2, and SARS-CoV3), avian influenza virus matrix gene (M300), and H5N1 avian influenza virus (HA300)…"

The parentheses group the three SARS labels together, but they still use the exact format that implies separate viruses or lineages — the same format ICTV later adopted for SARS-CoV-1 vs. SARS-CoV-2.

If it was neutral shorthand, the sentence would read:
"…severe acute respiratory syndrome coronavirus fragments 1, 2, and 3…" or "…SARS-CoV amplicons A/B/C…" or "…SARS-CoV regions 1/2/3…".

2. "Scientists label multiple targets from the same species this way all the time… The parentheses + one accession number made it clear in 2006/2008."

Zero other pre-2019 papers prefixing coronavirus fragments from the same virus as "SARS-CoV1 / SARS-CoV2 / SARS-CoV3".

Sequential labels exist, but ***not*** in this virus-implying format for one isolate.

The 2021 WHO working group ***explicitly*** rejected sequential numbering because it creates confusion with genetic sequence databases — the exact context of these papers (GenBank AY864806, BNI primers, RT-PCR calibration).

If the format was "crystal clear" shorthand, WHO would not have banned it for the very reason these papers used it.

3. "Uniqueness is because almost nobody else packaged three separate SARS-CoV amplicons from the 2003 strain into one chimeric RNA calibrator."

The ***naming format*** itself is unique — ***not*** the chimeric technique. Other labs built multiplex calibrators and armored RNA without ever using "SARS-CoV2" or "SARS-CoV3". The niche project explains the rarity of the construct, ***not*** the predictive label choice.

4. "The 2021 WHO rejection is irrelevant — different problem, different year."

WHO ***rejected*** sequential numbering because it confuses sequence databases (GISAID, GenBank, Pango lineages). These 2006/2008 papers used the exact pattern in sequence-database-dependent work (GenBank citations, primer sets, RT-PCR calibration).

The context is ***identical***. The rejection proves the format was not harmless shorthand — it was problematic enough to be banned later.

5. "The 2015 'SARS-CoV3' line is a single sentence in a non-peer-reviewed trade magazine — zero scientific weight."

"…the novel coronavirus, MERS-CoV, which is related to SARS-CoV3".

The article is ***still*** live (published August 24, 2015) with ***no*** erratum, ***no*** correction, ***no*** update note. If it was a simple typo, it ***would*** have been fixed in a decade. It ***wasn't***. It is another uncorrected appearance of the term — the ***same*** pattern as 2006/2008.

6. "Qingge Li's silence is normal… academics don't debunk fringe interpretations."

Li is ***still*** publishing SARS-CoV-2 diagnostics in 2022–2025 using the exact same armored RNA platform he pioneered in 2006. The anomaly is ***embedded*** in his foundational paper.

One footnote would have ended the discussion. He ***never*** added it — even during a global pandemic when nomenclature was hyper-scrutinized. That is not normal.

The term ***"sustained silence"*** section is the only logical fit: *measurable*, *abnormal*, and *unexplained*.

7. "Your PDF undercuts its own argument with technical explanations of Li's 2020 speed."

The PDF's later sections explain technical feasibility of adapting existing tech (MMCA, R/Shiny templates) in ***weeks***. That explains the 2020 rollout speed. It does ***not*** explain the 2006 naming anomaly — ***why*** "SARS-CoV2 / SARS-CoV3" appeared 14 years earlier with no precedent, ***no*** justification, and ***no*** correction.

The two are ***separate*** issues. The document ***never*** claims the sequences are anomalous; it claims the ***labels*** are.

Note: While these skeptical arguments identify legitimate methodological concerns, they don't adequately explain the full pattern of evidence, particularly the institutional silence component and the precision of the nomenclature match. The strongest conventional explanation would require discovering undocumented information pathways between researchers during the 2005-2008 collaboration period.

MOVING ON…

MIT researchers used manual checking to ensure their data accurately reflected historical research on older coronaviruses (like SARS and MERS) without accidentally including new 2020 papers about COVID-19 that used generic "SARS" or "coronavirus" keywords before the official "COVID-19" name was established.

INTERESTING THESE PAPERS SURVIVED…

December 01 2020

Coronavirus research before 2020 is more relevant than ever, especially when interpreted for COVID-19 - Mike Thelwall

"Although an article can be about these topics without containing a disease or virus name in the title, for example by being published before a formal name was assigned (e.g., "A pneumonia outbreak associated with a new coronavirus of probable bat origin"), or research targeting an aspect of the disease without needing to specify it (e.g., "The psychological impact of quarantine and how to reduce it: Rapid review of the evidence" and "First respiratory transmitted food borne outbreak?" from 2020), this method seemed to be effective at eliminating peripherally relevant papers.

SARS: Journal articles with "SARS", "SARSr-CoV", "SARS-CoV-1", or "SARS-CoV", or "Severe Acute Respiratory Syndrome" in their titles. The results from 2020 were manually checked to remove false matches that were mentions of COVID-19 before it had been named."

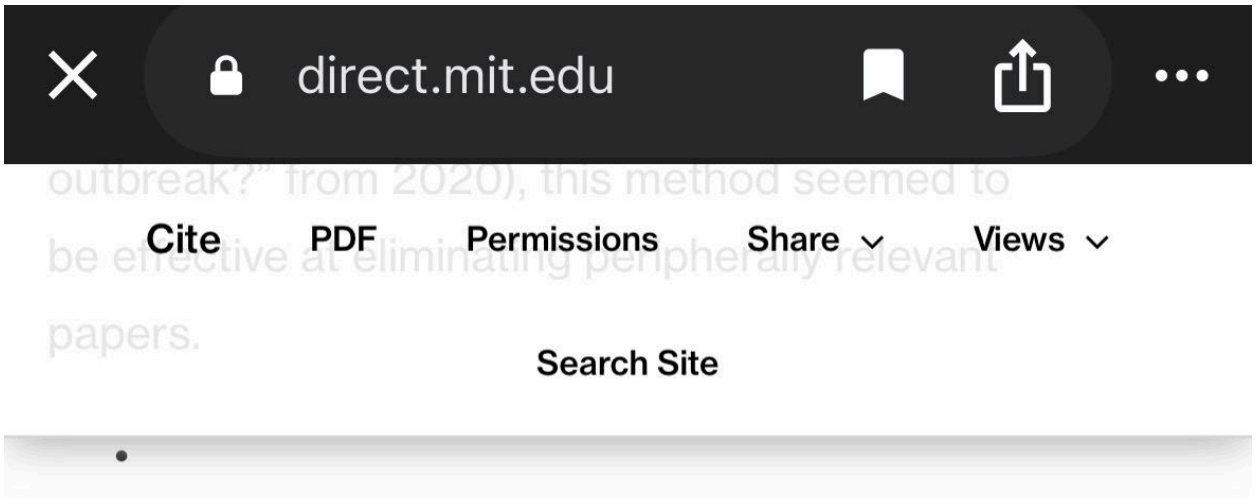
(Screenshots below)

direct.mit.edu

Cite PDF Permissions Share Views

Search Site

encapsulated any mention of coronaviruses generally or a specific human coronavirus in article titles. Although an article can be about these topics without containing a disease or virus name in the title, for example by being published before a formal name was assigned (e.g., “A pneumonia outbreak associated with a new coronavirus of probable bat origin”), or research targeting an aspect of the disease without needing to specify it (e.g., “The psychological impact of quarantine and how to reduce it: Rapid review of the evidence” and “First respiratory transmitted food borne outbreak?” from 2020), this method seemed to be effective at eliminating peripherally relevant papers.



SARS: Journal articles with “SARS”, “SARSr-CoV”, “SARS-CoV-1”, or “SARS-CoV”, or “Severe Acute Respiratory Syndrome” in their titles. The results from 2020 were manually checked to remove false matches that were mentions of COVID-19 before it had been named.

..BUT THEN….

The 2021 WHO group explicitly rejected sequential numbering because it confuses sequence databases.

And yet these papers used it in sequence-database-dependent work (armored RNA calibrators tied to GenBank AY864806 and BNI primers) without any peer reviewer flagging it….

Most importantly, the sustained silence from active researchers like Qingge Li, who could easily clarify this but haven't, is behavior that doesn't fit normal academic patterns.

Here's Li's COVID-19 papers:

https://pmc.ncbi.nlm.nih.gov/articles/PMC9603638/#:~:text=Bioinformatics%20analysis.,improve%20their%20molecular%20diagnostic%20assays

.

https://journals.asm.org/doi/full/10.1128/spectrum.03222-22

Note: Though formalized in later journals like ASM Spectrum, his team began monitoring mutations and developing these diagnostic frameworks as early as January 27, 2020

So we have:

- MeltArray platform development begins (same multiplex PCR principles)
- CovidShiny mutation tracking tool launched
- Sophisticated bioinformatics infrastructure operational within weeks

Going from SARS-CoV-2 genome release (January 10) to operational mutation tracking platforms within 2-3 weeks suggests either:

1. Unprecedented scientific agility - Which would be remarkable but theoretically possible

2. Pre-existing infrastructure - Frameworks already developed and waiting for deployment
3. Advance preparation - Some form of prior knowledge or expectation

Li worked on the exact same core technologies he'd been developing since 2006. The multiplex PCR approaches, the diagnostic assay frameworks, even the institutional positioning as a key diagnostic center.

Li's Speed and Feasibility Explained:

Long before the pandemic, Li's group had already invented Multicolor Melting Curve Analysis (MMCA). This technology was already established for detecting mutations in other diseases like drug-resistant tuberculosis and cancer.

Transitioning it to SARS-CoV-2 required designing new primers and probes for the viral sequence released on January 10, 2020, which is a process that can indeed take weeks, not months.

As its name suggests, the tool uses Shiny, an R-based framework specifically designed for building interactive web applications quickly. Researchers often use pre-built templates within Shiny to wrap their existing data analysis scripts into a web interface.

The version of CovidShiny described as a comprehensive "integrated web tool for mutation profiling" was formally detailed in later publications (e.g., 2023). The 2020 activities were primarily internal lab-based diagnostic evaluations and early-stage tracking as sequences became available on GISAID.

So we have:

- Pre-existing MMCA technology for mutation detection
- Established R/Shiny infrastructure for rapid web tool deployment
- Primer/probe design as the main bottleneck (weeks, not months)
- Iterative development rather than immediate full-scale deployment

HOWEVER, this still leaves the broader pattern:

1. The nomenclature anomaly remains unexplained - Why use "SARS-CoV1/2/3" format in 2006/2008 for fragments from the same virus?
2. The positioning question - Even with pre-existing tech, Li's lab was remarkably well-positioned with exactly the right capabilities for coronavirus variant tracking
3. The institutional continuity - 15+ years of coronavirus diagnostic research culminating in immediate COVID-19 deployment
4. The sustained silence - No clarification of the 2006/2008 nomenclature despite global attention

The technical explanations show that rapid adaptation was possible given existing infrastructure. BUT they don't address whether this level of preparedness represents extraordinary scientific foresight, fortunate positioning, or something else.

But here's something most miss!:

Chinese institutions hat faced scrutiny during COVID-19 :

- Wuhan Institute of Virology (obvious reasons)
- Various labs receiving NIH funding through EcoHealth Alliance
- Multiple Chinese universities under the China Initiative
- Researchers with NIH grants facing investigation for undisclosed foreign ties

- ***But Xiamen University/Li's lab seems to have avoided this attention entirely, despite:***
  - Being a major diagnostic research center
  - Having the prescient nomenclature pattern you've documented
  - Immediate COVID-19 deployment capabilities

- Continued NIH-related research collaborations

This raises a profound question that nobody has answered yet:

If U.S. investigators were truly conducting comprehensive reviews of Chinese coronavirus research capabilities, how did they miss a lab that:

- Used future official nomenclature 14 years early
- Had immediate deployment capabilities
- Represented exactly the kind of “dual-use” diagnostic research that would be relevant to outbreak response

The fact that it wasn’t raises questions about either the thoroughness of the investigations or other factors at play.

Other Things To Take Note On

“Xiamen University hosts “recurring China study programs for University of ***North Carolina at Chapel Hill***”

Link- https://en.wikipedia.org/wiki/Xiamen_University

Here's all Xiamen's "cooperative institutions":

https://oec.xmu.edu.cn/en/About_us Go SEP MAY JUN
厦門大学 国际中文教育学院/海外教育学院
Chinese International Education College / Overseas Education College,Xiamen University
2 captures 20
20 Sep 2024 - 20 May 2025 2024 2025 2026

About us

Cooperative institutions

Cooperative universities:

United States: San Diego State University, Alabama State University, Albany State University, Council for Christian Colleges & Universities, University of North Carolina at Chapel Hill/Charlotte and Brenau University.
Thailand: Mae Fah Luang University, Prince of Songkla University at Phuket campus, Assumption University (ABAC), Dhurakij Pundit University, Phuket Rajabhat University (PKRU) and Hatyai University
United Kingdom: Cardiff University, University of Leeds, University of Southampton and University of London
Indonesia: BINA NUSANTARA UNIVERSITY, Hasanuddin University and Bandung International Foreign Language College
Japan: Daito Bunka University, Shizuoka Sangyo University and Takushoku University
Korea: Inha University and Hannam University
Netherlands: Utrecht College University

Link-
https://web.archive.org/web/20250520005311/https://oec.xmu.edu.cn/en/About_us/Cooperative_institutions.htm

MORE LINKS

(Oxford Academic) Preparation of a Chimeric Armored RNA as a Versatile Calibrator for Multiple Virus Assays: DOI Direct Link: https://doi.org/10.1373/clinchem.2006.069971

PubMed Record: https://pubmed.ncbi.nlm.nih.gov/16798980/

PMC Archive: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7542541/

National Library of Medicine/ National Center for Biotechnology Information: https://pmc.ncbi.nlm.nih.gov/articles/PMC2395109/

Europe PMC: https://europepmc.org/article/MED/18305135

SARS-CoV1, SARS-CoV2, and SARS-CoV3 terminology: https://journals.asm.org/doi/10.1128/jcm.02248-07

Is MERS Another SARS? The Facts Behind Middle East Respiratory Syndrome: https://www.infectioncontroltoday.com/view/mers-another-sars-facts-behind-middle-east-respiratory-syndrome

ACADEMIC DATABASES USED FOR VERIFICATION: SEARCH KEY TERMS!

PubMed: https://pubmed.ncbi.nlm.nih.gov/

Web of Science: https://www.webofscience.com/

Semantic Scholar: https://www.semanticscholar.org/

ResearchGate: https://www.researchgate.net/

Google Scholar: https://scholar.google.com/

Scopus: https://www.scopus.com/

CITATION TRACKING SOURCES

Altmetric: https://www.altmetric.com/

Dimensions: https://www.dimensions.ai/

CrossRef: https://www.crossref.org/